The precise pathogenesis of retinal arterial macroaneurysms is not well known and the vessel abnormalities that precede their development have rarely been demonstrated. Focal arterial wall damage is the likely precursor of retinal artery macroaneurysm. Macroaneurysm formation from the exact site of a previous retinal artery embolus has been described in only four cases including the present one.[6–8] It is postulated that local vessel wall damage from emboli predisposes to aneurysmal dilatation. The incidence of carotid atheromatous plaques in patients with retinal artery macroaneurysms was higher than that found in a similar asymptomatic hypertensive population, supporting the theory that retinal artery macroaneurysms may be of embolic origin.[9] Cardiovascular evaluation of the patient reported here revealed a congenital atrial septal defect; this may have caused retinal arterial embolic episodes, leading to focal arterial wall damage and development of macroaneurysms.

Most macroaneurysms exhibit vascular leakage resulting in retinal oedema, and the secondary accumulation of lipid exudate. The oedema may be due to direct aneurysmal leakage or leakage from small incompetent vessels surrounding the aneurysm.[2,10] These microvascular changes resemble the reorganisation of small vessel networks which have been shown in experimental embolic arterial occlusions.[11] Palestine *et al.*[10] recommended photocoagulation to the small, incompetent vessels surrounding the aneurysm (which contribute to retinal swelling). It is generally agreed that the visual prognosis is poorer in patients who experience visual loss from extensive macular oedema or exudate than in patients with decreased vision secondary to macular or vitreous haemorrhages.[2,3] In the case described here, resolution of haemorrhage, and serous macular detachment with improvement in visual acuity, followed yellow wavelength photocoagulation treatment of the leaky, telangiectatic blood vessels surrounding the macroaneurysm.

The author thanks Ms Connie B. Unisa-Marfil for her secretarial work.

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Sir,

Ultrastructural analysis of opacities seen in a hydrophilic acrylic intraocular lens

Intraocular lens (IOL) optic opacities have been observed in foldable acrylic IOLs. In some patients they can cause glare, reduced visual acuity and contrast sensitivity. In this article we present an electron microscopic study of opacities in an explanted hydrophilic acrylic IOL.

Case report

A 67-year-old patient was referred to our department in December 1998 because of progressive visual loss in her left, pseudophakic eye. The cataract operation was performed in January 1998 in another clinic. According to the surgical report, the operation was uneventful. A one-piece acrylic IOL was implanted: SC60B-OUV (DGR Incorporated, St Petersburg, FL).

Slit lamp biomicroscopy of the left eye revealed a clear cornea and no inflammation in the anterior chamber. The IOL was in the capsular bag. The IOL optic was opaque due to many tiny whitish opacities. Both haptics were clear. Visual acuity (VA) was 20/400, IOP was 12 mmHg. The IOL was explanted, and exchanged for a PMMA IOL.

The IOL was analysed with a Philips PSEM 500 scanning electron microscope (Philips, Eindhoven, Holland), a Zeiss EM902 transmission electron microscope (Zeiss, Oberkochen, Germany) and a Hitachi H600 transmission electron microscope (Hitachi, Dusseldorf, Germany) equipped with a Kevex 7000 energy-dispersive X-ray microanalysis system (Getac, Mainz, Germany).

The IOL optic was opaque due to many tiny whitish opacities (Fig. 1). Both haptics were clear, as was the edge of the IOL optic. On scanning and transmission electron

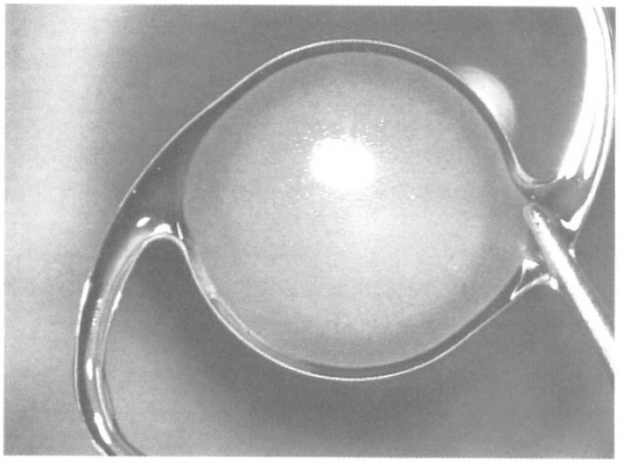

Fig. 1. *The explanted IOL (intraoperative view immediately after explantation – picture taken from video).*

microscopy the IOL displayed a smooth surface. Transmission electron microscopy showed opacities of different size (ranging from 2 μm to 15 μm) starting about 20 μm beneath the IOL surface. High-power magnification showed a crystalline structure of the opacities (Fig. 2, inset A). The electron diffraction image showed the inorganic nature of the crystals. Energy-dispersive X-ray microanalysis showed that the crystals consisted of calcium, phosphorus and oxygen – presumably calcium phosphate (Fig. 2, inset B). The copper represents artifact from the preparation of the specimen.

The IOL was left to dry for 2 months and the microscopic study repeated. The intensity of the IOL opacities did not change.

Comment

IOL optic opacities have been observed in PMMA[1] and foldable acrylic IOLs.[2] Discoloration of silicone IOLs has also been reported.[3] Both discoloration and glistenings have been attributed to the hydration of the IOL optic material. In acrylic IOLs it has been postulated that glistenings are caused by vacuoles of water in the acrylic optic visible because of the refractive index differences.[2,4] Our findings are not consistent with the hypothesis that the opacities are related to hydration of the acrylic IOL material, although this may reflect differences between the acrylic materials.

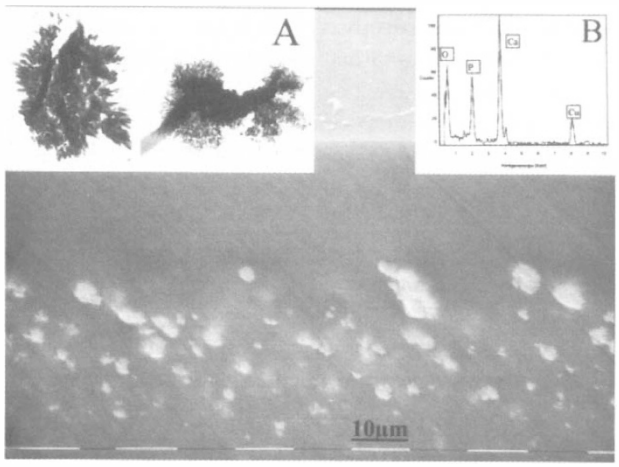

Fig. 2. *Main picture: Scanning electron micrograph of the IOL optic cross-section (×1400). Inset A: High-power transmission electron micrograph of the opacities (×14 000). Inset B: Energy-dispersive X-ray spectrum of the opacities.*

It is remarkable that the opacities were localised about 20 μm beneath the IOL surface and spared the rim and the haptics of the IOL. We do not have any plausible explanation for such a distribution except that it could be related to the manufacturing process of the IOL. The cataract operation was performed in January 1998 in another clinic. According to the surgical report, the operation was uneventful. The fine details of the surgical procedure could not be obtained. It is unlikely that the viscoelastic substance or infusion fluid contained an excessive level of calcium since commercially available products were used. If calcium-containing products were the cause of the IOL opacities one would have expected the IOL to have been opacified immediately after surgery. According to the 1 month post-operative slit lamp examination the IOL was clear at this time.

If the opacification of the IOL is due to a process similar to dystrophic calcification, it would be expected to occur only in patients with abnormal serum calcium and phosphate ion levels. Our patient did not have any sign of parathyroid abnormalities. The serum calcium and phosphate ion levels were normal.

Previous reports found that glistening in acrylic IOLs appeared after their hydration and that dissipation of the glistening occurs when the lenses are removed from the water bath and dried. In our case the IOL opacities did not change after drying for 2 months. The opacities in our case consisted of inorganic crystals containing calcium, phosphorus and oxygen – presumably calcium phosphate.

Proprietary interest: None.

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Sir,

Eyelid skin atrophy associated with chronic usage of ophthalmic steroid ointment, and its successful treatment with the Versapulse laser

Long-term topical corticosteroids are used with caution in the eye for fear of their potential side-effects such as raised intraocular pressure and cataract formation. Skin atrophy is a well-documented side-effect of the long-term use of dermatological preparations of corticosteroids, related to the thickness of the skin to which they are applied, the potency of the steroid and the duration of its use. We describe a patient who sustained dermatological eyelid change following the use of an ophthalmic steroid preparation, and who was then successfully treated by novel means.

Case report

A 55-year-old woman presented to the eye clinic complaining of a 5 year history of recurrent 'bruised eyes', often upon waking. She stated that prominent blood vessels had developed within her lower eyelid skin and that extensive bruising of the lids frequently occurred with minimal, if any, physical contact. There was no history of trauma or atopy. In response to direct questioning, she disclosed that for more than 30 years she had been applying Betnesol (Evans) ophthalmic ointment, obtained on repeat prescription from her general practitioner, onto her eyelids as treatment for blepharitis.

On examination, the lower eyelid skin was atrophic with underlying prominent, dilated blood vessels and visible orbital fat. There was a mild degree of anterior blepharitis but no other signs of skin or ocular disease (Fig. 1). A diagnosis of corticosteroid-related skin atrophy was made. In addition to cessation of ointment use, treatment options included direct cauterisation and therefore closure of the vessels, or excision of the affected skin together with its underlying vessels. It was felt, however, that neither would result in optimum cosmesis: the former might scar the overlying skin, whilst without the addition of a skin graft, with the latter there would be a significant risk of ectropion. It was considered, however, that the use of a 'vascular' laser to produce light of a wavelength that would be highly absorbed by haemoglobin might result in vessel closure with minimum risk of complications.

Treatment was therefore subsequently undertaken using the Coherent Versapulse cutaneous laser, producing light at 532 nm. Using a 'chilled tip' on the laser probe to cool the skin in order to minimise epidermal damage, the lower eyelid skin was treated at a fluence of 155/cm^2 and pulse width of 20 ms. As expected there was a considerable degree of post-laser inflammation, but this settled within 3 weeks of treatment. On review 2 months after treatment, the blood vessels in question within the lower eyelids were no longer visible and the symptoms completely resolved. In addition, somewhat unexpectedly, there was also a significant improvement in the general appearance of the eyelid skin, with a return to near-normal texture and colour (Fig. 2).

Comment

Eyelid skin atrophy induced by topical ophthalmic corticosteroids has only been reported on one previous occasion.[1]

Epidermal side-effects of topical steroid use include thinning of the epidermis and cessation of proliferation in the basal layer. Dermal side-effects include the inhibition of elastin and type I and III collagens, by a reduction in collagen mRNA synthesis which decreases directly the strength of the underlying dermis.[2] Blood vessels also become increasingly fragile due to the decreased support by collagen and glycosaminoglycans.[3] Long-term vasodilatation can occur, hence the development of telangiectases. Rebound inflammation may also occur with acute cessation of topical steroid use.

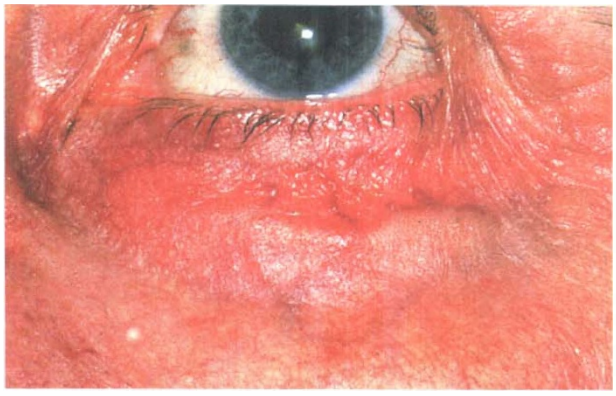

Fig. 1. *The lower eyelid on presentation, showing underlying prominent, dilated blood vessels and visible orbital fat.*

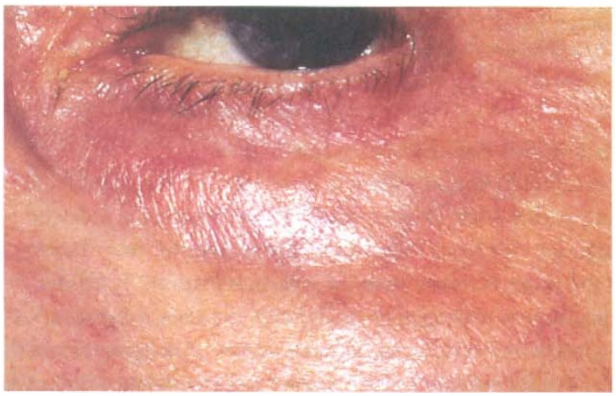

Fig. 2. *The lower eyelid after treatment with the Versapulse laser, showing a near-normal appearance.*